Danielle Ferstler -

This past summer, I spent four weeks at the Fox Chase Cancer Center (FCCC) in Philadelphia, conducting breast cancer research invited by world renowned scientists Drs. Jose and Irma Russo. I was awarded this opportunity through the Huntington Breast Cancer Action Coalition (HBCAC) Students and Scientists Summer Research Program. The entire experience was incredible and I feel very fortunate to have had the opportunity to work with these dedicated scientists and the staff of FCCC.

The research I conducted at the FCCC Breast Cancer Research Laboratory was designed to study the effect of 17β -estradiol on human breast epithelial cell proliferation (growth). 17β -estradiol is the most potent estrogen in the human body. Estradiol affects not only the uterus and breast, but also the brain, liver, and male reproductive organs. It has been shown in previous studies that estradiol has very high estrogenic activity during reproductive years. Estrogens induce responses in hormone sensitive organs (i.e. the breast) through estrogen-receptors, and that combination (along with a ligand) activates gene transcription, or the copying of genes. Previous research studies have shown that estrogens help initiate cancer progression. There is evidence to suggest that estrogens initiate uncontrolled gene transcription of cancer cells.

For the experiment, I tested three concentrations of 17β-estradiol on bsMCF cells, a cell line considered to be cancerous and invasive, similar to metastasized cancer cells. The cells are derived from the breast epithelium (tissue cells) of a woman who donated her breasts after being diagnosed with a disease wherein her breasts formed noncancerous solid masses. These cells are called MCF-10F cells. For the purposes of this lab, the bsMCF cells are cancer cells and the cells which they originate from are normal cells. In reality, the MCF-10F cells are not normal because they formed benign masses; however, they are the "most normal" cells that have been used to trace the progression of breast cancer.

Each day, I would place fresh media, the cells' food, with the different concentrations of 17β -estradiol in the cells. I also added Alamar Blue, a stain that is used to indicate the viability of living cells. Alamar blue is a cell permeable compound called resazurin. Living cells continuously reduce the resazurin, which is blue in color, to resorufin, which is red in color. After adding the Alamar Blue, the cells were incubated for four hours and then placed in the Epoch Reader, which uses light rays to measure the proliferation of the cells. I confirmed that the cells had grown by observing that the Alamar had changed color from blue to a purple-pink. Also, I looked at the cells under the microscope, and observed that there were more cells each day.

For all three weeks of the experiment, I used blank cells, control cells, and cells with one of the three different concentrations. The blank cells had no concentrations or Alamar Blue. (The blank cells were needed for the Epoch Reader to do its calculations, along with confirming that the Alamar Blue has no effect on the cells-which it did not.) The control had Alamar Blue but no concentrations. For the first week, I treated the cells with the three concentrations of 17β -estradiol for two days and I only used the Alamar Blue on the last day of treatment. Therefore, data was only collected once, and the results signify both days of treatment together. For

the next two weeks the cells were treated with three concentrations of 17β -estradiol for three days each week and I used the Alamar Blue each day. The data I collected demonstrated how the cell proliferation changes after 24, 48, and 72 hour treatments.

The results indicated that the concentrations of 17β -estradiol I used did not produce a statistically significant effect on the cell proliferation of bsMCF cells. A "statistically significant" result would mean that the proliferation of the cells was caused by the 17β -estradiol, and not by other factors. My results are not significant for several possible reasons. The incubator I was using in the lab was malfunctioning; the CO₂ levels were above what they were supposed to be for a healthy environment for the cells. It is likely that the excess CO₂ was killing the cells, decreasing the number of cells and the amount of proliferation. Also, the treatment periods were short. Perhaps if I left the concentrations in for 96 hours or 120 hours, the 17β -estradiol would have had a significant effect.

Although the data was not significant, that does not mean the experiment or the experience was insignificant. Any type of result shows something, and that something is more knowledge to help make improvements for the next set of experiments. One important lesson I learned was that research does not always go as planned. To me, this means that you have to have the ability to overcome things that are out of your control utilizing your intellectual and creative skills. This past summer at the FCCC has taught me how to push my limits in order to overcome the bumps in the road and reach my ultimate goal of completing the experiment. I would especially like to thank my mentors: Dr. Jose Russo, Dr. Irma Russo, Dr. Julia Pereira, Maria Barton, David Armiss, and Yubo Zhai for their guidance and support through this endeavor. This certainly is an experience I will never forget.